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Case report

***Shewanella putrefaciens* infective endocarditis**



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ABSTRACT

Shewanella putrefaciens rarely causes infection in humans. In the last few decades a growing number of cases have been described. The following report outlines the case of a 40-year-old immunocompetent white man with *S. putrefaciens* infective endocarditis. This is the first known case of infective endocarditis due to an apparently monomicrobial *S. putrefaciens* infection, and the second known case of *S. putrefaciens*-related infective endocarditis worldwide.

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Introduction

Shewanella spp. encompass a group of saprophytic Gram-negative oxidative bacilli generally present in warm climates as part of marine microflora.¹ Other possible sources include aquatic reservoirs, oil and gas reserves, as well as soil, snake bites, fish, poultry, dairy and beef products.² Although apparently abundant in the environment, human infection with these pathogens is relatively rare. When identified in human isolates, it usually presents as a mixed bacterial flora.² Of the *Shewanella* spp., human clinical infections are caused by *Shewanella algae* and *Shewanella putrefaciens*, the greater share of which being the more pathogenic *S. algae*, possibly due to its ability to carry out beta-hemolytic type reactions and exotoxin production.² The most common clinical manifestations are skin and soft tissue infection, bacteremia and otitis.^{3,4} *Shewanella* spp. have also been associated with abdominal, joint and bone, pulmonary, urinary, ophthalmic, and cerebral infection.^{1,4} Bacteremia is often present but the course is

usually benign.^{1,4} There is only one known case of polymicrobial infective endocarditis related to *S. putrefaciens*.⁵ The following report outlines the first known case of infective endocarditis due to an apparently monomicrobial *S. putrefaciens* infection, and the second known case of *S. putrefaciens*-related infective endocarditis worldwide.

Case report

A 40-year-old immunocompetent man presented to his vascular surgeon with complaints of right hand swelling, calor, numbness, and pain. An upper extremity venous doppler showed thrombosis of the right radial vein. He was admitted to the hospital and started on anticoagulation therapy with heparin. Two weeks prior to admission the patient presented with a burn at the medial region of his right thigh, which was evaluated by his primary care physician and topical antibiotics were prescribed. He had also been complaining of shaking chills for several days prior to admission. Past medical his-

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tory was significant for seizure disorder, dyslipidemia, tobacco use, asthma, bronchitis, sleep apnea, hypertension, hypothyroidism, osteoarthritis, right carpal tunnel release surgery, and venous insufficiency. Physical examination revealed a 4/5 systolic heart murmur over mitral valve area, a 7 cm × 5 cm ulcer over medial region of the thigh with surrounding erythema secondary to burn and edema, erythema and calor involving the right thumb extending into the wrist.

On hospital day (HD) 2, the patient started having intermittent fever (every 48 h) up to 103.5 °F with shaking chills. Blood cultures were obtained; however, no antibiotics were initially prescribed as no clear infectious source was identified. On HD 7, a venogram of right upper extremity showed resolution of the venous thrombosis. Cefazolin 1 g intravenously every 8 h was empirically started on the same day for persistent fever. He was noted to have an elevated procalcitonin level (40 ng/mL), C-reactive protein (7.1 mg/dL), erythrocyte sedimentation rate (65 mm/h) and rheumatoid factor (15.5 IU/mL). White blood cell counts were within normal limits. On HD 8, right hand erythema and pain symptoms worsened, warranting repeated ultrasound evaluation of right upper extremity, which revealed the presence of thrombus in the distal radial artery. The persistent fever, the presence of systolic murmur, and thrombo-embolic phenomenon involving the radial artery led to investigation of possible endocarditis with transesophageal echocardiography revealing a 1.6 cm × 1.0 cm vegetation in the posterior mitral valve leaflet with hypoechoic areas suggestive of valvular abscess. Antibiotic was changed to Cefepime 2 g intravenously every 12 h and Gentamicin 120 mg intravenously every 8 h for the treatment of endocarditis. Two sets of blood cultures (each with aerobic and anaerobic bottles) were taken throughout the hospital stay on HDs 4, 7 and 12. Eventual growth of Gram-negative bacilli was detected on all six sets of blood cultures [BacT/ALERT blood culture system (bioMérieux, Durham, NC)]. The Gram-negative bacilli were identified by VITEK 2 automated system (bioMérieux, Durham, NC), as *S. putrefaciens* with 99.9% probability. Patient was transferred to a tertiary care center and had a valvectomy of the posterior leaflet of mitral valve. Pathology of mitral valve leaflet showed granulation tissue with acute fibrinous inflammation. The valve cultures were negative. Subsequently, the patient was lost to follow-up.

Discussion

Despite the vast prevalence of *Shewanella* spp. in the environment, they are rarely implicated in clinical scenarios as a source of pathogenicity.⁴ Two species of *Shewanella* (*S. algae* and *S. putrefaciens*) are known to occasionally cause infection in humans. Khashe et al.² reported that certain phenotypic characteristics could be useful to differentiate *S. putrefaciens* from *S. algae*. Compared to *S. algae*, *S. putrefaciens* is nonhemolytic on sheep blood agar, unable to grow at 42 °C or on high salt media (6.5%), but able to produce acid from arabinose, maltose, and sucrose.² Automated identification systems have been reported as not being able to distinguish *S. algae* from *S. putrefaciens* reliably, as *S. algae* has not been updated in the databases.¹ Khashe and Janda² tested five strains of *S. algae* and five strains of *S. putrefaciens* on different

bacterial identification systems that included API 2OE, API NFT, RapID NF Plus and the automated VITEK system. All of these identification systems misidentified *S. algae* as *S. putrefaciens*. However, manual reading of arabinose and maltose reaction permitted the distinction between *S. algae* and *S. putrefaciens*. Despite the utility of the production of acid from carbohydrate oxidation for the correct identification of *S. algae*, *S. putrefaciens* can produce varied results which have led to the recommendations of performing 16S rRNA sequencing for the correct speciation of *Shewanella* spp.³ In our case, the identification of the microorganism was performed through VITEK 2 system. The new VITEK 2 system has now included in its database *S. algae* and theoretically it could be detected on clinical samples. As the accuracy of the identification of *S. algae* on the VITEK 2 system, to our knowledge, has not been reported, it is possible that this strain could have been misidentified as *S. putrefaciens* instead of *S. algae*.

S. putrefaciens, when detected, is often a component of polymicrobial infections.⁴ This type of presentation has made it difficult to determine the significance of *S. putrefaciens* as an isolated pathogen. However, *S. putrefaciens* monomicrobial infections have been reported, confirming the pathogenicity of this microorganism. Vignier et al.⁴ reported that half of their cases of *Shewanella* infection were monomicrobial. In their review of the literature, they determined that a skin or mucosal portal of entry was found in 53% of the reported *Shewanella* infection cases. These were often preceded by chronic ulceration of the lower limb, trauma, burn wound, or seawater exposure. In our patient, the source of *S. putrefaciens* was not identified. Nonetheless, it is reasonable to assume that the patient's burn wound was the port of entry. Unfortunately, no cultures of the burn wound on the patient's right lower limb were obtained.

Dhawan et al.⁵ described a case of *S. putrefaciens* and viridans group streptococci polymicrobial mitral valve endocarditis in a patient with known rheumatic heart disease. Compared to our case, only one microorganism was isolated validating the pathogenic role of *S. putrefaciens*. Six sets of blood cultures, in a period of 9 days, turned out positive for *S. putrefaciens* resulting in the confirmation of monomicrobial infection due to *S. putrefaciens*. This rare case of monomicrobial infective endocarditis by *S. putrefaciens* in an immunocompetent host adds to the current literature of the increased numbers of human infection associated with the organism and highlights the pathogenic role of *Shewanella* spp.

Conflicts of interest

The authors declare no conflicts of interest.

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